

# Human ECG changes during prolonged hyperbaric exposures breathing N<sub>2</sub>-O<sub>2</sub> mixtures

JAMES M. WILSON, PAUL D. KLIGFIELD, GEORGE M. ADAMS,  
CLAUDE HARVEY, AND KARL E. SCHAEFER

Naval Submarine Medical Research Laboratory, Groton, Connecticut 06340

WILSON, JAMES M., PAUL D. KLIGFIELD, GEORGE M. ADAMS, CLAUDE HARVEY, AND KARL E. SCHAEFER. *Human ECG changes during prolonged hyperbaric exposures breathing N<sub>2</sub>-O<sub>2</sub> mixtures*. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 42(4): 614-623, 1977. — In an effort to determine whether hyperbaric exposures while breathing N<sub>2</sub>-O<sub>2</sub> mixtures have an effect on cardiac depolarization and repolarization, electrocardiograms of 10 divers participating in four N<sub>2</sub>-O<sub>2</sub> saturation dives were analyzed. In all cases, a decline in heart rate was observed upon compression to saturation depth (20-30%); a slow adaptation and return of heart rate toward normal was observed in those dives where the depth and environmental parameters remained constant. Whenever excursion dives were performed, the heart rate responded by decreasing on deeper excursions and increasing on upward excursions. Hyperbaric bradycardia disappeared after 8 days at pressure during the saturation dives at 50 and 60 feet seawater gauge (fswg), but was still present at this time at 200 fswg. The magnitude of the hyperbaric bradycardia produced by excursion dives following saturation at depth was influenced by the state of adaptation of heart rate. Decompression was uniformly accompanied by a rapid increase in heart rate resulting in a significant elevation in the postdive period. Alterations in myocardial repolarization as evidenced by Q-T interval, ST, and T wave changes were observed. Development of slight right ventricular conduction delay compatible with right ventricular strain was noted in four of the divers during the two deepest dives to 100 and 198 fswg. During the latter dive, progressive decrease in P wave amplitudes and eventual loss of P waves resulting in an apparent nodal rhythm was observed in one diver. Multiple premature ventricular contractions occurred in another diver. These observations, along with the reports by other authors, suggest that the different variables associated with the hyperbaric environment—gas density, pressure, inert gas—have a definite effect on the pacemaker activity of the heart and myocardial depolarization and repolarization.

saturation diving; hyperbaric air; heart rate

EXTENSIVE STUDIES characterizing the changes in the electrical activity of the human heart during apneic immersion diving have been reported (4, 10, 19, 26, 28, 33). However, limited information is available concerning electrocardiographic variations during nonapneic diving, in particular, during prolonged residence in a hyperbaric chamber or underwater laboratory as in a saturation dive.<sup>1</sup> During these dives, mixtures contain-

ing oxygen diluted with nitrogen, helium, or neon are the principle gaseous constituents. Recent studies have suggested a direct effect of these gas mixtures on neurological tissues (24). The effect of the gases on cardiac conduction tissues under saturation conditions is, for the most part, unknown. A few authors (19, 26, 28) have mentioned the occurrence of P wave, rhythm, ST and T wave changes, while focusing on the well documented bradycardia associated with increased ambient pressure (4, 10, 28). A detailed analysis of the electrocardiographic changes that have occurred during saturation exposures has not been reported.

The present study characterizes the electrocardiographic findings that were observed during four saturation dives. Though some of the changes suggest subclinical alterations in both pacemaker activity and myocardial depolarization and repolarization, patterns have emerged that are clinically significant.

## MATERIALS AND METHODS

The electrocardiograms from four saturation dives involving 10 U.S. Navy divers have been analyzed. All subjects were graduates of the Naval School of Diving and Salvage and had extensive prior operational diving experience. None of the divers had a history of cardiopulmonary symptoms. Extensive predictive evaluations that included pulmonary functions, blood analyses, numerous electrocardiograms, exercise tolerance studies, and physical examinations substantiated this history.

Each of the dives was carried out in the hyperbaric chamber complex at the Naval Submarine Medical Research Laboratory in Groton, Connecticut. Three of the four dives were in the Shallow Habitat Air Dive (SHAD) program, while the other dive was the first of the Nitrogen Saturation (NISAT) series. Figure 1 describes the pressure profiles and Table 1 delineates the environmental parameters of each dry, simulated dive.

SHAD-I was a 30-day compressed air dive to a saturation depth of 50 feet seawater gauge (fswg) or 2.515 atmospheres absolute (ATA), involving two subjects. Excursions were made upward to 5 fswg (1.512 ATA) and downward to 235 fswg (8.121 ATA) for varying lengths of time. The dive was hyperoxic with the oxygen tension at saturation depth being 0.51 ATA. Standard six lead (I, II, III, AVR, AVL, AVF) electrocardiograms

<sup>1</sup> A saturation dive is an exposure at increased ambient pressure of sufficient duration at depth to permit the attainment of equilib-

rium between the ambient gaseous environment and the gases dissolved in the divers' tissues.

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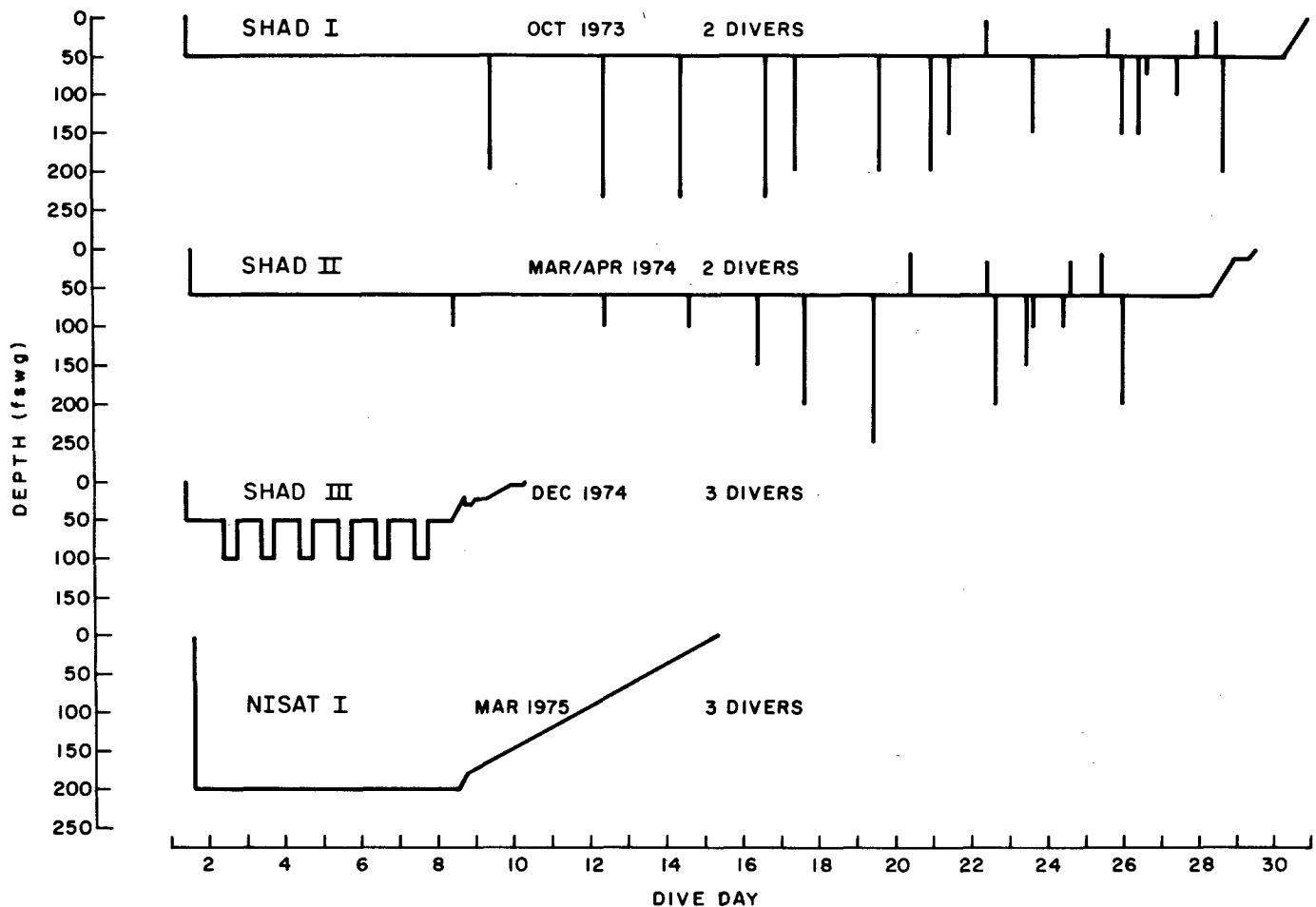


FIG. 1. Dive profiles.

TABLE 1. *Environmental parameters*

Residence depth				
fswg	50	60	50	198
ATA	2.515	2.818	2.515	7.000
Total duration, days	29.5	28	9	14.25
Mean O <sub>2</sub> level, ATA	0.51	0.57	0.61	0.297
Mean CO <sub>2</sub> level, %, surface, equiv	0.092	0.099	0.172	0.125
Mean temp, °F	76.8	73	75.8	76.5
Mean relative humidity, %	62.0	52.0	74.7	59.9
Excursion profile				
No. of ascending excursions	4	4	0	0
Depth of shallowest excursion				
fswg	5	5	0	0
ATA	1.512	1.512	0	0
No. of descending excursions	14	11	6	0
Depth of deepest excursion				
fswg	235	250	100	0
ATA	8.121	8.576	4.030	0

were performed at saturation depth at approximately the same time of day after the last excursion or at approximately the same time of day on a day of rest.

In SHAD-II, two subjects lived at a depth of 60 fswg (2.818 ATA) for 29 days; during this period excursions were made upward to 5 fswg (1.512 ATA) and downward to 250 fswg (8.576 ATA). This dive was hyperoxic, the mean oxygen tension equaling 0.57 ATA. Standard six lead (I, II, III, AVR, AVL, AVF) electrocardiograms were taken each day at approximately the same time of

day at saturation depth, after the last excursion, or on a day of rest.

SHAD-III, involving three subjects, was an 8-day compressed air dive to 50 fswg (2.515 ATA) with 8-h excursions to 100 fswg (4.030 ATA) each day. This hyperoxic dive had a mean O<sub>2</sub> tension of 0.61 ATA. Twelve lead electrocardiograms (I, II, III, AVR, AVL, AVF, V<sub>1-6</sub>) were performed at the same time of day at excursion depth and upon return to saturation depth. A rhythm strip (lead 2) was taken during compression to saturation depth.

In NISAT-I, three subjects lived and worked at a depth of 198 fswg (7.000 ATA) for 7 days, followed by a 7-day decompression period. No excursion dives were performed. This was a relatively normal oxygen tension dive, the breathing media being composed of 97.03% nitrogen and 2.97% (0.23 ATA) oxygen. Twelve-lead (I, II, III, AVR, AVL, AVF, V<sub>1-6</sub>) electrocardiograms were performed each day.

The electrocardiograms were recorded on either a Cambridge model VS-III American Optical Corp. Cardio Tracer or a Hewlett-Packard model 1500B electrocardiograph to obviate any change due to the differences in machines. Paper speed was 25 mm/s. Lead placement was constant, the sites having been marked before the dive with indelible ink. Anatomical placement conformed to standard clinical practice (6). All recordings were obtained in the resting state with the subjects in the supine position. Tracings were examined as to

rhythm, rate, QRS vector, T vector, P-R interval, QRS duration, Q-T interval, P wave, Q wave, ST segment, T wave, and voltage changes as compared to the 1-mv calibration signal. The intervals and wave durations documented represent averages of the respective values to avoid any error introduced by respiratory variations. Corrections for rate of the Q-T interval were calculated for each cardiogram using the formulae-corrected Q-T interval ( $Q-T_c$ ) =  $Q-T/\sqrt{R-R}$  (5). Vector analysis was performed using the trigonometric method (6).

Statistical analysis of the data was performed using the Student *t*-test. *P* values less than 0.05 were interpreted as being significant.

In SHAD-I and II the subjects routinely carried out exercise tests at 100 and 150 W lasting for 10 min each, at time intervals of approximately 1 wk. In SHAD-III the same exercise tests were performed every 2 days. No change in exercise tolerance was observed. Since the high pressure and gas density in NISAT-I (7 ATA) was expected to cause a marked increase in the resistance to breathing, the work load was reduced from 150 to 125 W. However, the reduction in work load did not prevent a  $CO_2$  accumulation and reduction in ventilation during exercise at a work load of 125 W at 7 ATA pressure. Although the general conditions during NISAT-I have to be considered as stressful, there was no significant "stress response" inasmuch as blood cortisol was unchanged and urinary ketosteroid excretion was only slightly increased. Under these circumstances, it is unlikely that an unspecific stress effect had a significant influence on the observed ECG changes.

## RESULTS

For descriptive purposes, each of the SHAD dives will be divided into three phases: phase 1, representing the period of compression to saturation depth and equilibration; phase 2, the excursion period; and phase 3, the period of decompression and postdive recovery.

**SHAD-I.** No significant change occurred in P waves, P-R interval, rhythm, QRS or T vector, QRS duration, ST segment, T waves, or voltage during SHAD-I.

Heart rate (Fig. 2) declined significantly ( $P < 0.01$ ) upon compression to saturation depth. This decrease from  $70.00 \pm 1.89$  beats/min pre-dive to  $56.00 \pm 5.65$  beats/min remained at approximately the same level for 2 days; on *dive day 3* a slow return toward pre-dive values began; pre-dive values were reached on *dive day 8*. At the beginning of phase 2, on *dive day 9*, the first excursion caused a significant decline in heart rate ( $P < 0.01$ ). This pattern of significant depression of heart rate on deep excursions continued throughout the excursion period. The upward excursion on *dive day 22* caused a slight but significant ( $P < 0.05$ ) elevation in mean heart rate. Decompression (phase 3) began on *dive day 30* and with it an increase in heart rate that terminated in a very significant ( $P < 0.01$ ) tachycardia in the early postdive period. On *postdive day 1* the mean heart rate was  $93.00 \pm 4.24$  beats/min with a return to pre-dive values not occurring until *postdive day 3*.

The corrected Q-T interval ( $Q-T_c$ ) showed a pattern similar to heart rate (Fig. 2). During phase 1, the interval was shortened from a pre-dive mean of  $0.412 \pm 0.008$  s to  $0.260 \pm 0.000$  s upon compression to saturation depth. On *dive day 5* a slow increase in the  $Q-T_c$  began which continued until phase 2 was started. In phase 2, the deep excursions caused significant shortening ( $P < 0.01$ ) of the  $Q-T_c$ . All values obtained after excursion dives and on rest days were significantly depressed ( $P < 0.05$ ) from pre-dive levels except on *dive day 22* (upward excursion) when the  $Q-T_c$  was not significantly different from pre-dive values. Beginning with phase 3 a rapid increase in the  $Q-T_c$  began, resulting in a significant ( $P < 0.05$ ) elevation of the  $Q-T_c$  on *dive day 30* and *postdive days 1* and 2. As with heart rate, the greatest elevations occurred on the days immediately after the dive ended; a return to pre-dive values occurred on *postdive day 3*.

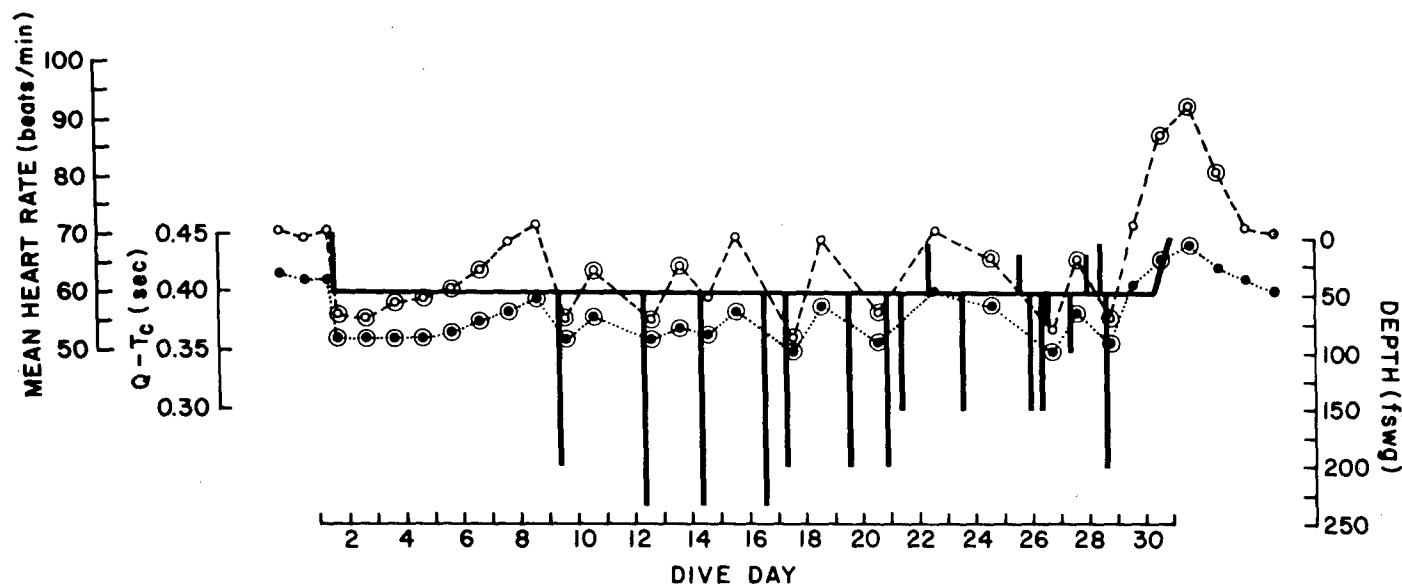


FIG. 2. SHAD I. Mean heart rate (○—○) and mean  $Q-T_c$  (●—●) are superimposed over dive profile to show correlation with changes in depth. Values statistically different from controls at 5% level and better are marked by second circle.

**SHAD-II.** During SHAD-II, significant change in P waves, P-R interval, QRS or T vector, QRS duration, ST segment, T waves, or voltage did not occur. Rhythm changes were not observed; all tracings demonstrated normal sinus conduction.

During phase I the mean heart rate (Fig. 3) declined from  $69.66 \pm 1.86$  beats/min predive to  $53.50 \pm 2.12$  beats/min ( $P < 0.01$ ) upon compression to saturation depth. This decrease remained constant until *dive day 4* when a slow return toward predive levels occurred. Predive levels were reached on *dive day 7* and on *dive day 8* phase 2 began. As in SHAD-I, the deep excursions caused significant depressions in heart rate ( $P < 0.01$ ) while upward excursions resulted in elevations of heart rate. On *dive day 20* when a solitary upward excursion was made, the mean heart rate was  $71.00 \pm 1.41$  beats/min. Although this was not significantly different from predive levels, it was different ( $P < 0.01$ ) from the mean of the previous day. With the onset of decompression (*dive day 28*) a rapid increase in mean heart rate occurred resulting in a very significant tachycardia ( $P < 0.01$ ) on the day the dive ended and on *postdive days 1* and *2*. A return to predive levels occurred on *postdive day 3*.

The corrected Q-T interval (Fig. 3) declined from a predive mean of  $0.398 \pm 0.008$  to a mean of  $0.355 \pm 0.007$  s ( $P < 0.01$ ) during phase 1. On *dive day 3* a slow increase in the Q-Tc began with a return to predive levels observed on *dive day 7*. During phase 2, the deep excursions resulted in a significant depression ( $P < 0.01$ ) of the Q-Tc while the isolated upward excursion (*dive day 20*) resulted in a Q-Tc increase to predive levels. During phase 3, the Q-Tc increased to significantly elevated levels ( $P < 0.01$ ) on the last dive day and *postdive day 1*. A return to predive levels occurred on *postdive day 3*.

**SHAD-III.** No significant change in P waves, P-R interval, QRS or T vector, QRS duration, ST segment, or voltage occurred during SHAD-III. With the first

excursion on *dive day 2*, the tracings of all three men demonstrated slight right ventricular conduction delay characterized by rSR' complexes in the right precordial leads and diphasic T waves (Fig. 4). The recording machine and leads were changed to obviate any error; however these diphasic patterns persisted until *dive day 4* in two subjects and *dive day 5* in the third subject. The only arrhythmias observed during this dive were the occasional premature atrial contractions noted during exercise tolerance testing at maximum work load (150 W).

Since excursions began on *dive day 2* phase 1 lasted only one day (Fig. 5). The predive mean heart rate was  $72.88 \pm 3.62$  beats/min; rhythm strips taken during compression to saturation depth showed an initial increase in mean heart rate to  $91.00 \pm 3.62$  beats/min (not shown in Fig. 5). A few minutes after saturation depth had been obtained, the mean heart rate fell to  $58.33 \pm$

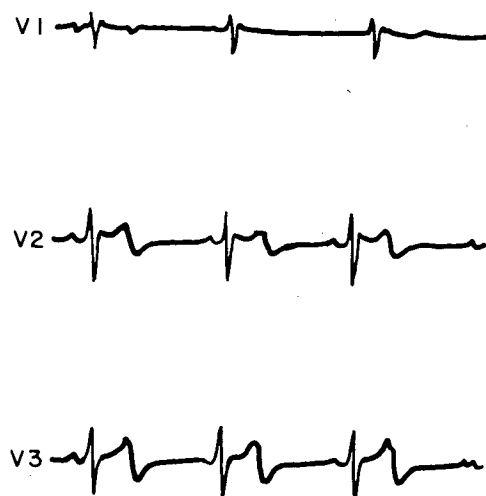


FIG. 4. Tracing on diver RO during SHAD-III at 100 fswg; rSR' complexes in right precordial leads and diphasic T waves are evident.

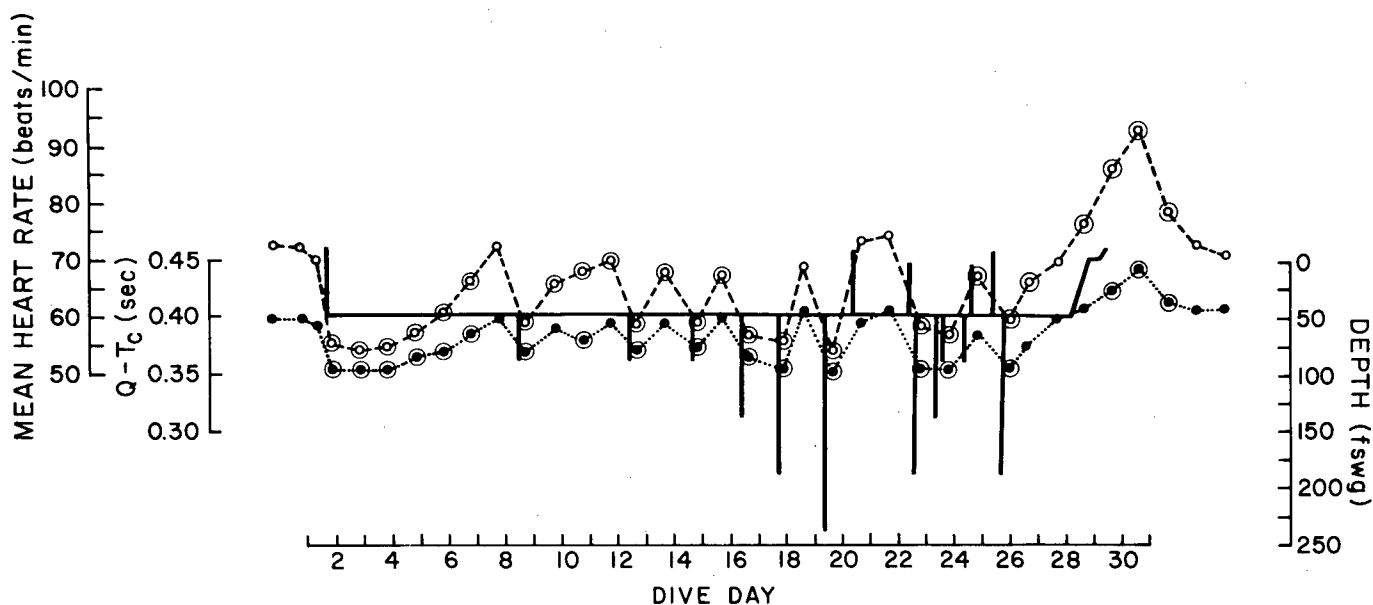


FIG. 3. SHAD II. Mean heart rate (○—○) and mean Q-Tc (●—●) are superimposed over dive profile as in Fig. 2. Values

statistically different from controls at 5% level and better are marked by second circle.

2.51 beats/min ( $P < 0.01$ ). During phase 2 the first excursion resulted in a further depression of the heart rate to a mean of  $53.00 \pm 0.00$  beats/min ( $P < 0.01$ ). A return to 50 fswg did not cause a significant change from the heart rate taken at 100 fswg. However, the excursions on subsequent days caused significant depressions in mean heart rate ( $P < 0.01$ ) from the predive levels with elevations (as compared to the mean heart rate at excursion depth) of mean heart rate after return to saturation depth. A general trend toward predive levels can be seen beginning on *dive day 3*. Upon return to 50 fswg on *dive day 7*, the mean heart rate reached predive levels. Phase 3 began on *dive day 8* with an elevation of mean heart rate resulting in a very significant ( $P < 0.01$ ) tachycardia on *dive day 10* (surface) and the first postdive day. Equilibration to predive levels occurred on *postdive day 3*.

The corrected Q-T intervals demonstrated the same pattern as in SHAD-I and II. The mean Q-Tc (Fig. 5) declined from  $0.41 \pm 0.005$  s on the surface to  $0.37 \pm 0.006$  s upon compression to saturation depth ( $P < 0.01$ ). During phase 2, the Q-Tc decreased significantly ( $P < 0.01$ ) with each excursion, returning to an increasingly elevated level as phase 2 progressed. With the onset of decompression, the mean Q-Tc began to increase, resulting in a very significant ( $P < 0.01$ ) elevation above predive levels on *dive day 10* (surface) and *postdive day 1*. A return to predive levels occurred on *postdive day 2*.

*NISAT-I*. During the first 8.5 h, the mean oxygen concentration was 0.23 atm (174.8 Torr); at the same time divers JB and NT were symptomatic, experiencing severe lethargy, slurred speech, difficulty in breathing, and vomiting. The third diver did not report any symptoms during this time period. The electrocardiograms of *diver JB* during the first few hours showed a lengthening of the P-R interval (from 0.14 s predive to 0.21 s) to the upper limits of normal for that heart rate. The P waves became progressively more flattened in appearance with eventual loss of the P waves resulting in an

apparent nodal rhythm (Fig. 6). Electrocardiograms obtained in the afternoon began to show minor ST elevations in the lateral precordial leads with subsequent tracings demonstrating the development of slight right ventricular conduction delay characterized by rSR' complexes in the right precordial leads; a diphasic T wave pattern was also apparent (Fig. 7). The oxygen concentration was then increased to 0.31 ATA (235 Torr); after 8 h, the rSR' complexes and T wave changes disappeared (Fig. 7) and a change in voltage, QRS and T wave configuration was evident. The P waves were more demonstrable after the oxygen was added, but a flattened appearance persisted.

The electrocardiograms on the other symptomatic diver (NT) showed occasional premature ventricular contractions and peaking of the T waves in leads  $L_3$  and  $V_3$  (Fig. 8). After oxygen was added, the premature ventricular contractions disappeared and a minor change in QRS configuration was noted (Fig. 8). The tall, peaked

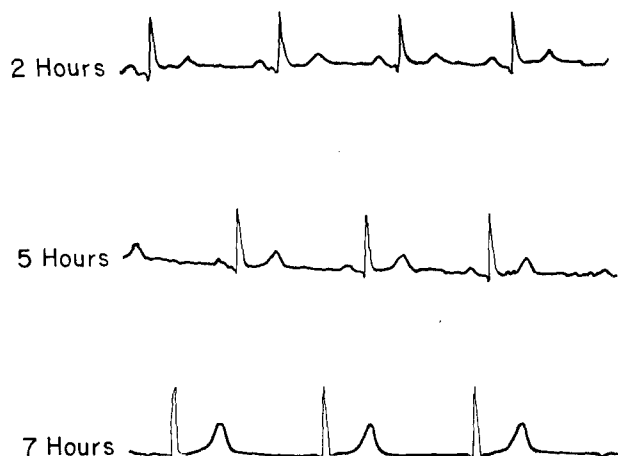


FIG. 6. Lead II tracings on *diver JB* during NISAT-I showing an apparent progressive loss of the P waves. Tracings taken 7 h after saturation depth (198 fswg) was reached show loss of P waves with no retrograde P waves visible.

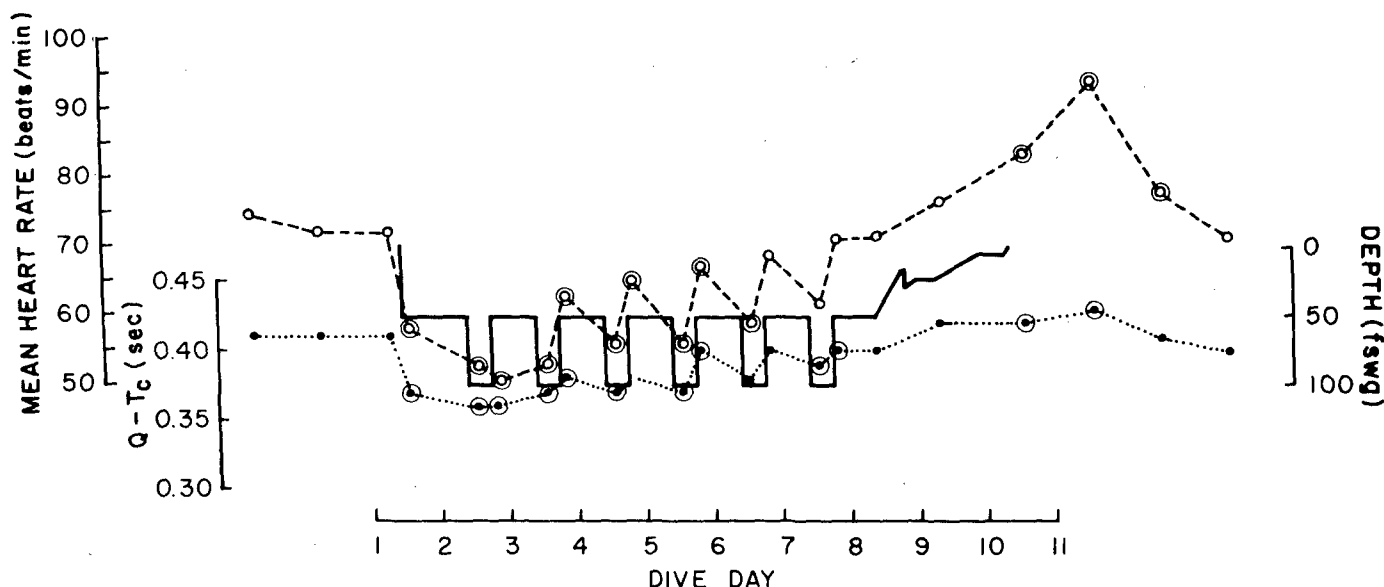


FIG. 5. Dive profile of SHAD-III with mean heart rate (○—○) and mean Q-Tc (●·····●) superimposed. Values statistically different from controls at 5% level or better are marked by second circle.

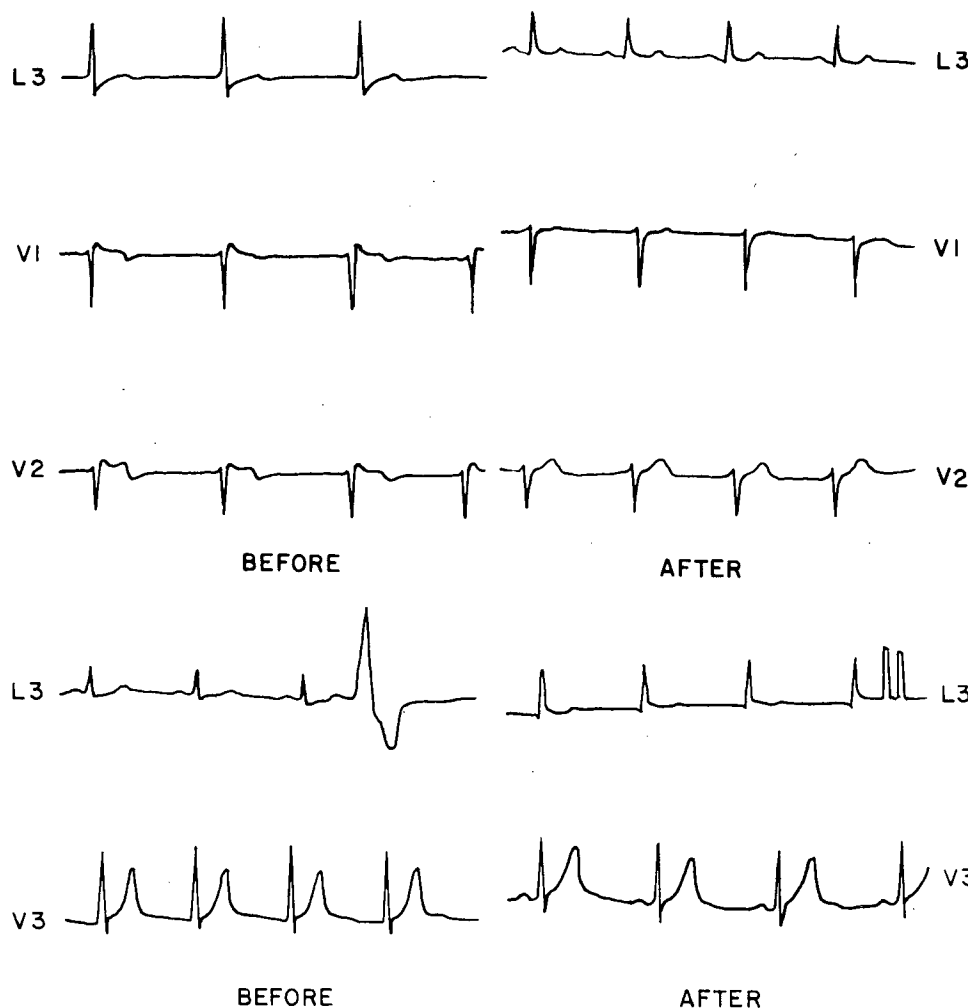


FIG. 7. Tracings on *diver JB* during NISAT-I at 198 fswg before oxygen concentration was increased. Note flattened appearance of P waves, rSR' complexes in the right precordial leads, and diphasic T waves. Tracing on *diver JB* approximately 8 h after oxygen concentration was increased. Note disappearance of rSR' complexes in V<sub>1</sub> and diphasic T waves in V<sub>2</sub>.

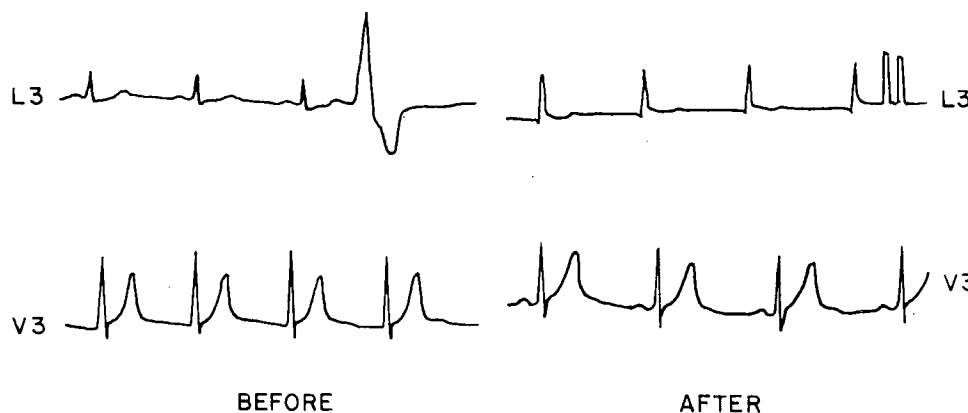


FIG. 8. Tracing on *diver NT* during NISAT-I at 198 fswg before oxygen concentration was increased. Note premature ventricular contractions in L<sub>3</sub> and the tall T waves in V<sub>3</sub>. Approximately 8 h after oxygen concentration was increased, premature ventricular contractions had disappeared, while tall T waves persisted.

T waves in the precordial leads persisted throughout the dive.

The electrocardiograms on the one diver who was asymptomatic (*RJ*), showed no significant alterations after the oxygen concentration was increased and no change throughout the dive.

The only arrhythmias observed were during exercise tolerance testing at maximum work load (125 W). Premature atrial contractions were noted in *divers NT* and *JB*; one of the divers (*NT*) experienced episodes of premature ventricular contractions during exercise tolerance testing.

The heart rate (Fig. 9) declined from a mean of  $70.88 \pm 3.10$  beats/min to a mean of  $48.00 \pm 2.00$  beats/min ( $P < 0.01$ ) upon compression to saturation depth. A further decline to  $45.66 \pm 1.52$  beats/min was observed on *dive day 2*. On *dive day 3*, the mean heart rate increased slightly and continued to increase slowly throughout the dive. Toward the end of the decompression (*dive day 11*) the mean heart rate reached predive levels and continued to increase to a mean of  $83.00 \pm 2.64$  beats/min ( $P < 0.01$ ) on the day of surfacing and  $99.33 \pm 5.13$  beats/min on the first postdive day ( $P < 0.01$ ). A return to predive levels occurred on *postdive day 3*.

The Q-Tc (Fig. 9) declined from a mean of  $0.407 \pm 0.01$  s to  $0.353 \pm 0.006$  s ( $P < 0.01$ ) upon compression to saturation depth. It decreased further to  $0.343 \pm 0.006$  s

( $P < 0.01$ ) on *dive day 2*. The Q-Tc began a sustained increase on *dive day 3*. On the day the divers surfaced (*dive day 15*) the mean Q-Tc was  $0.437 \pm 0.006$  s ( $P < 0.01$ ) and was  $0.443 \pm 0.006$  s ( $P < 0.01$ ) on *postdive day 2*. Adjustment to predive levels was observed on *postdive day 3*.

#### DISCUSSION

The decline in heart rate associated with increased ambient pressure has been documented in other studies. It has been observed in simulated human dives in air (32), oxygen (27), nitrogen-oxygen mixtures (3), and helium-oxygen mixtures (31). Flynn et al. (15) have shown that both pressure and gas density have an effect on heart rate. Increased oxygen tensions (21, 32) are also thought to play a role. No attempt was made in the present study to determine which parameters were causing the observed effects.

From the data presented, it is apparent that each of the dives demonstrated the same general pattern of changes in heart rate. A very significant depression of heart rate was observed on compression to saturation depth. SHAD dives I, II, and III were hyperoxic (mean oxygen concentrations of 0.51, 0.57, and 0.61 ATA, respectively) while NISAT-I was relatively normoxic (oxygen concentration equal to 0.297 ATA). When oxygen

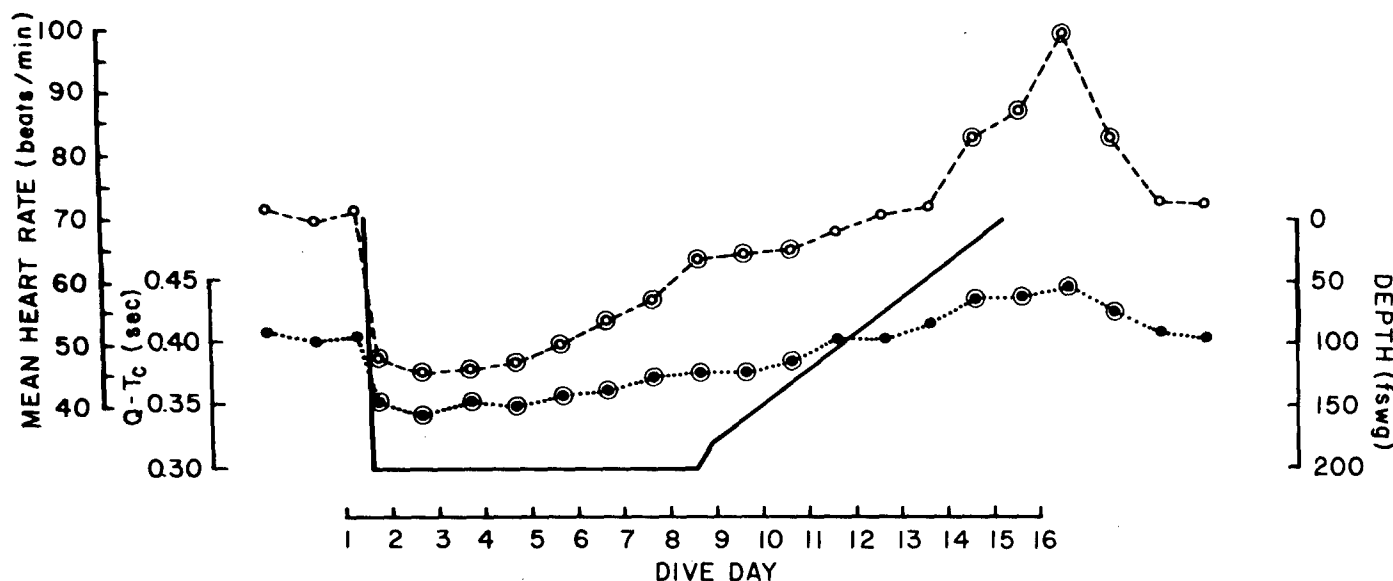


FIG. 9. NISAT-I, Dive profile with mean heart rate (○—○) and mean Q-Tc (●·····●) superimposed. Values statistically significant

cantly different from controls at 5% level and better are marked by second circle.

was added to the atmosphere during the first 8 h in NISAT-I, the heart rate decreased further from  $48.00 \pm 2.00$  beats/min to  $45.66 \pm 1.52$  beats/min. Albano (3) and Whalen (32) have also observed a further decline in heart rate when the oxygen concentration was increased at depth.

Flynn et al. (15) in studies involving the helium-oxygen environment, have suggested that while the changes in heart rate during relatively shallow dives (less than 300 fswg) are dependent upon pressure and gas density, the depression becomes fixed and relatively independent of pressure and density for deeper dives. Morrison et al. (25) have found that in the helium-oxygen environment the extent of the bradycardia did not change significantly beyond 600 fswg.

In SHAD-I and II and NISAT-I, a slow increase in the mean heart rate began 2-3 days following compression to saturation depth. Thus, while the depth and environmental parameters remained constant, the heart rate slowly returned toward normal. As shown in Table 2, the time required for the heart rate to adapt to hyperbaric conditions and to return to control values was 8 days in SHAD-I and SHAD-II. In NISAT-I heart rate remained significantly below initial values at day 8. Daily excursion from 50 to 100 fswg in SHAD-III did not change the adaptation period of 8 days. In NISAT-I a greater depressant effect on heart rate occurred and adaptation had not been reached at the end of the saturation dive on day 8. Whether this attenuated equilibration was due to the greater pressure, gas density, or higher nitrogen tension in NISAT-I remains unknown. Q-Tc interval changes closely paralleled those of heart rate.

The heart rate did not become insensitive to further changes in pressure upon its return to predive levels at depth. In the SHAD dives, the first excursions brought about very significant depressions in heart rate as did subsequent excursions. Upward excursions resulted in an elevation of heart rate.

TABLE 2. Adaptation time of heart rate and Q-Tc interval following initial response to hyperbaric exposure breathing  $N_2O_2$  mixture

	Experimental condition saturation depth		Average heart rate, min				Average Q-Tc interval, s			
	fswg	ATA	control	day 1	day 7	day 8	control	day 1	day 7	day 8
SHAD I	50	2.515	69.7	53.5	64.0	70	0.400	0.355	0.385	0.400
SHAD II	60	2.818	70.0	56.0	69.0	72	0.410	0.360	0.385	0.395
NISAT	198	7.00	70.8	48.0	57.0	63.7	0.407	0.353	0.373	0.377
SHAD III	50 ↑ 100	2.5 ↑ 4.0	72	58.3	69.7	71.3	0.410	0.37	0.380	0.400

TABLE 3. Influence of adaptive state on responses of heart rate and Q-Tc interval to excursion dives to 100 fswg from a saturation depth of 60 fswg

Exptl condition	Average heart rate, min				Average Q-Tc interval, s			
	control	prior	end	Δ	control	prior	end	Δ
Surface	70				0.410			
Saturation depth 60 fswg								
Excursion 60 → 100 fswg								
Day 8	70	65.5	-13.5		0.400	0.360	-0.040	
Day 12	67.5	56.6	-11.0		0.395	0.370	-0.025	
Day 14	65.5	56.5	-9.0		0.395	0.375	-0.020	

The state of adaptation of heart rate did influence the response to subsequent excursions, as can be seen in Table 3. The magnitude of pulse rate reduction following excursion dives to 100 fswg from a saturation depth of 60 fswg depends on the level of heart rate prior to the excursion dive. In the first case, in which a full adaptation had occurred and the control heart rate values measured at the surface had been reached, the fall in heart rate produced by the excursion dives to 100 fswg was maximal. In subsequent excursion dives, in which the initial values were lower than surface control values, a smaller decrease in heart rate was observed. The Q-Tc interval changes followed in the same direction.

Adaptive changes in the heart rate requiring shorter time have been noted in a hyperbaric helium environment (5.48 ATA) in which the relative bradycardia disappeared after 10 h of exposure (9). Moreover, in a 24-day dry heliox saturation dive to 18.6 ATA, hyperbaric bradycardia disappeared after 3 days at pressure and reappeared after 13 days at pressure (29). The difference in the adaptation time of heart rate between nitrogen-oxygen and helium-oxygen dives cannot be explained at this time.

Hamilton et al. (17) and Albano (3), using helium-oxygen mixtures, have observed an initial tachycardia on compression with a final bradycardia after a few minutes at depth. We have observed the same effect using air in SHAD-III. Rhythm strips taken during compression to saturation depth demonstrated initially elevated heart rates followed by a bradycardia after a few minutes at depth. This is probably the effect of anticipation of compression and hyperventilation with a resultant increase in sympathetic discharge on the heart. Once the divers had reached saturation depth and resting electrocardiograms were taken, the bradycardia was evident.

Whether the bradycardia was due to changes in oxygen concentration (3, 32), pressure (7, 15), or cardiopulmonary reflexes from changes in gas density (23) could not be determined from the present study. Isolated guinea pig heart studies (14) have shown that heart rate and myocardial contractility are unaffected by exposure to hyperbaric conditions. It would thus seem that the alterations observed are due to changes in the autonomic control of the heart. Indeed, Daly and Bondurant (12) and Albano (3) have abolished the bradycardia associated with breathing air and oxygen at depth with injections of atropine. The effects of pressure on parasympathetic and sympathetic stimulation are not known. Akers and Carlson (1) have postulated, from studies involving smooth muscle preparations, that pressure alters the cholinergic receptor response, thus increasing the time required for acetylcholine to interact with the receptor. Fagraeus (14) has presented evidence that high inert gas pressure and/or hydrostatic pressure per se interfere with the sympathetic stimulation of the heart. Additional studies on pacemaker activity and myocardial conduction in the hyperbaric environment are required.

With the beginning of decompression and recovery, all four divers showed an increase in heart rate resulting in a very significant elevation in the heart rate in the postdive period. The highest elevation of mean heart rate occurred 24 h after surfacing. All of the divers, except for the men involved in the shorter SHAD-III dive, complained of dyspnea on exertion in the early postdive periods. Hamilton et al. (17) and Albano (3) have observed gradual increases in heart rate during decompression. Hamilton (16), Korotayev (20), and Akhlamov (2), have observed a similar elevation in heart rate during the postdive period. Whether this rebound elevation is due to physical deconditioning from many days of relative inactivity (16), to a removal of parasympathetic inhibition, an increase in sympa-

thetic activity, a combination of these, or unknown factors has remained obscure.

The occurrence of rSR' complexes in SHAD-III and NISAT-I represents a delay in right ventricular conduction and is consistent with mild right ventricular strain. The etiology of this delay in conduction is uncertain. It was observed in one diver in the first few hours of NISAT-I and disappeared after the oxygen concentration was increased. This would suggest that hypoxia resulting in constriction of the pulmonary vasculature and pulmonary hypertension was the cause of the right ventricular strain pattern. However, the same right ventricular conduction delay was observed in all three divers in SHAD-III, a dive with a rather high oxygen concentration. Whether the conduction delay was the result of hypoxia in NISAT-I, adjustment to the hyperoxia in SHAD-III, breathing gases at greatly increased densities and pressure, disordered myocardial electrolyte flux, or a combination of these factors cannot be determined from the present study. These observations are consistent with those recently made by Dr. Ian Caldor (personal communication) who found evidence at autopsy of mild ventricular hypertrophy and pulmonary vasculature changes consistent with pulmonary hypertension in young professional British divers (less than 26 yr old) dying of unrelated causes. Further studies on the response of the pulmonary vasculature to hyperbaric conditions should be rewarding.

The Q-Tc changes followed the same pattern in all the dives as the heart rate changes. In all the dives the Q-Tc shortened as the heart rate lengthened and vice versa. Of particular interest is the trend during decompression and postdive recovery period at a time when the heart rate was significantly elevated, the Q-Tc was significantly lengthened. These observations are unusual in that the Q-T interval should be relatively constant once it is corrected for heart rate.

Although a change in the T wave vector was not observed, a change in configuration of the wave was obvious. More subtle changes in vector direction might have occurred but the analysis method used was not sufficiently sensitive to discern them. Vector cardiographic studies with loop analysis of the P, QRS, and T waves such as those recently performed at Duke University by Dr. Horace Argeles (personal communication) will be most helpful in elucidating the changes in electrical activity. In this study, they found a lengthening of the Q-Tc upon compression breathing helium-oxygen mixtures with a shortening of the Q-Tc upon decompression. When they shifted to air during decompression, they observed a further shortening of the Q-Tc and the greatest T wave configuration changes. As was observed in these studies, hyperbaric air was related to a shortening of the Q-Tc and a change in the configuration of the T wave.

The repolarization changes and the right ventricular conduction delay observed in SHAD-III and NISAT-I are consistent with disordered myocardial electrolyte influx. In particular, increased calcium levels would account for the Q-Tc changes observed. It should be noted, however, that no clinically significant shift in



serum Na, K, Ca, or Mg occurred during these dives. Hyperbaric membrane transport studies should shed some light on the changes in membrane potential occurring under these varied conditions.

Albano (3) has observed a lengthening of the Q-Tc while using helium-oxygen mixtures and numerous authors have commented on T wave changes such as diphasic (20), flattened (2), and peaked (34) configurations using air and helium-oxygen mixtures. Slight ST elevations were observed in this study (NISAT-I) and the 198 fswg Genesis-E (heliox) dive (22).

It should be emphasized that the Q-Tc changes observed in SHAD-I, II, and III were all within the accepted clinical range for normal (0.35-0.44 s) (13). NISAT-I was the only dive in which the mean Q-Tc decreased below normal (0.34 s) and then only briefly.

No rhythm changes were observed during SHAD-I, II, or III. However, during NISAT-I some very interesting conduction aberrations were observed. Upon compression to saturation depth, the P-R interval of one man, JB, was prolonged to the upper limit of normal (0.21 s) for that heart rate. During the next few hours at depth, the P waves were flattened in all leads and by the end of the first 8 h, an apparent nodal rhythm was present, being manifested by loss of the P waves in all leads with no corresponding reduction in voltage. Retrograde P waves were not observed. This suggests that the depression of the normal atrial pacemaker became so intense that a lower nodal pacemaker escaped and became dominant before an intermittent or lower grade (1st or 2nd degree) block could develop. This may not have a true nodal rhythm but a sinus pacemaker in the absence of atrial discharge. Vassale and Greenspan (30) have shown experimentally that the sinus node can pace the ventricles, despite a lack of electrocardiographic evidence of atrial depolarization. Whatever the mechanism, the effect lasted only approximately 8 h. Tracings taken after the oxygen concentration was increased showed a normal sinus rhythm pattern, although the flattened appearance of the P waves persisted throughout the dive. Akhramov et al. (2) have observed a lengthening of the P-Q interval and compression of the P wave in the hyperbaric air environment and Hartmann et al. (18) have observed compression of the P waves and A-V nodal rhythms in the helium-oxygen environment.

The tracings on *diver NT* in NISAT-I showed occasional premature ventricular contractions during the first day of the dive. These were abolished when oxygen was added. On *dive day 6*, he became completely exhausted during an exercise tolerance test and failed to complete the study. A rhythm strip on *dive day 6* taken 15 min after the study showed occasional premature ventricular contractions. On *dive day 7* he became very tired during exercise tolerance testing and, when the work load was shifted to 125 W, his electrocardiographic tracing (trunk leads) demonstrated frequent premature ventricular contractions. The study was immediately terminated by the physician present. A full description of the arrhythmias observed during the exercise tolerance tests will be given in a separate communication. It

is interesting that only during NISAT-I were the major conduction disturbances observed.

It is thus apparent that NISAT-I was a stressful dive, showing not only the heart rate and Q-Tc changes observed in the earlier dives, but also evidences of conduction changes and possible myocardial irritability. It was, as mentioned, the deepest dive and the one with the highest tension of nitrogen. The gas density was also quite high, greatly increasing the work of breathing. It is interesting to note that the equivalent depth of a helium-oxygen dive based on gas density conversions would be approximately 1,550 fswg.

Arrhythmias in specific are rarely mentioned in the literature concerning saturation diving. In the SEALAB-II experiment (22), a 205-fswg helium/nitrogen/oxygen saturation dive, certain unspecified arrhythmias were observed; these were thought to be clinically insignificant. Korotayev et al. (20) observed evidence of ventricular extrasystoles during the air phase of the SADKO-II experiment.

Although apneic immersion diving is a notably different physiological stress from nonapneic dry chamber saturation dives, many of the electrocardiographic findings are similar. The bradycardia is without question a feature of the human diving reflex thought to be mediated via the vagus nerve (4, 10, 28). Flattening of the P waves, nodal rhythms, idioventricular rhythms, premature ventricular contractions, and peaked T waves (19, 26, 28) have been observed. Several authors have observed sinus tachycardia and atrial, nodal, and ventricular arrhythmias, including ventricular tachycardia in the immediate postdive period (26, 33). That the electrocardiographic changes observed during saturation diving are simply a modified diving reflex is conjectural at best. However, it is interesting that the pattern of changes is quite similar.

The standard clinical electrocardiogram is an inadequate tool for ascertaining the exact changes occurring in pacemaker activity and myocardial depolarization and repolarization. Isolated conduction tissue studies in the hyperbaric environment would be most rewarding and informative.

Some of the clinical implications of the present work are evident when reviewing the results of Criscuoli and Albano (11) and Brauer et al. (8). Criscuoli and Albano (11) found that when rats were exposed to increasing pressures breathing helium-oxygen mixtures, they progressed from sinus bradycardia to eventual A-V block with periodic asystole and finally to ventricular fibrillation and death. Brauer et al. (8) have observed that when squirrel monkeys were exposed to pressures greatly in excess of 100 ATA in the absence of anticonvulsant treatment, serious cardiovascular changes occurred resulting in eventual cardiac arrest. The depths involved in these two studies are in the range 100-150 ATA and obviously far deeper than man has ventured to date. However, in the future, man may be limited in his journey to the greater depths by a cardiovascular syndrome, just as he is apparently limited at present by a high pressure nerve syndrome (24). Investigations into the effects of the various hyperbaric conditions of gases,

pressure, and density on the pulmonary circulation, myocardial metabolism and electrolyte flux, and conduction tissue will be most helpful in elucidating the data presented herein.

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Address reprint request to: K. E. Schaefer, Biomedical Sciences Div., Naval Submarine Medical Research Laboratory, Box 900, Naval Submarine Base, Groton, Conn. 06340.

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